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GEOPHYSICAL VARIABLES AND BEHAVIOR: LXXXII. A STRONG ASSOCIATION BETWEEN SUDDEN INFANT DEATH SYNDROME AND INCREMENTS OF GLOBAL GEOMAGNETIC ACTIVITY—POSSIBLE SUPPORT FOR THE MELATONIN HYPOTHESIS[1]

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Summary.—A significant subpopulation of young infants who die suddenly (SIDS) often exhibit anomalies consistent with disruptions within the pineal-limbic system. We have hypothesized that sudden decreases in nocturnal melatonin by a specific range of geomagnetic activity would precipitate sudden infant death. A correlation of .90 was found between the numbers of cases of Sudden Infant Death Syndrome per month during the years 1960 and 1961 in Ontario and an increase (primarily) in numbers of days per month with average geomagnetic activity between 11 and 20 nT and 31 through 40 nT but a decrease in the numbers of days with values between 21 and 30 nT. This nonlinear sensitivity, presumably associated with specific ripple frequencies within the geomagnetic field, may explain the failure by other researchers to detect linear associations between the numbers of these types of deaths and gross, scalar indicators of geomagnetic activity.

Sudden Infant Death Syndrome (SIDS) is a major cause of death in human beings between one month and one year of age (Beckwith, 1988; Kinney, Filiano, & Harper, 1992; Little & Peterson, 1990; Murphy, Newcombe, & Sibert, 1982). Three-quarters of these mortalities occur between one and three months after birth. The dead infant is usually found between 2200 hr. and 0800 hr. local time. Approximately 70% of the cases occur during the autumn-winter months (Peterson, 1988; Peterson, Sabotta, & Strickland, 1988). According to Millar and Hill (1993), boys are more likely to be found dead than are girls (odds ratio 1.47). There may also be temporal clustering of SIDS within geographically large areas (Moreno, Ardanaz, Olivera, Castilla, & De Pedro-Cuesta, 1994; Rodrigues, Marshall, Murphy, & Osmond, 1992).

With the exception of the enhanced incidence of vomiting, apathy, and either hyper- or hypothermia, there are no obvious antecedent factors that distinguish infants who display SIDS from those infants who exhibit a general illness. Postmortem analyses have shown that babies whose deaths were attributed to SIDS exhibited anomalous carotid bodies (Naeye, Fisher, &

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Whalen, 1976), abnormal proliferation of astroglial fibers within the lateral reticular nucleus of the brain stem (Naeye, 1976a; Oehmichen, Linke, Zilles, & Saternus, 1989), aggregations of microglia (which implies tissue dysfunction at least more than about two to three days earlier) within the dentate gyrus of the hippocampus (Deligio & Becker, 1994), abnormal retention of brown adipose tissue (Lean & Jennings, 1989; Reid & Tervit, 1994; Valdes-Dapena, Gilane, & Catherman, 1976), hypertrophy of the chromaffin cells within the suprarenal glands, and petechial anomalies within the lungs.

The most frequent humoral anomalies include increased levels (Chacon & Tildon, 1981) of T3-thyroxine (117 ± 5.3 ng/ml for SIDS vs 62 ± 9.4 ng/ml for autopsy controls). Melatonin levels within the cerebrospinal fluid (Sturner, Lynch, Deng, Gleason, & Wurtman, 1990) have been significantly lower (91 ± 21 pg/l relative to 180 ± 27 pg/l) than for those dying from other causes. However, melatonin levels within the blood did not exhibit a significant difference. Increased levels of blood tryptase, a common inference of mast cell activity, have also been reported for these babies (Platt, Yunginger, Sekula-Perlman, Mirchandani, & Schwartz, 1994).

Weissbluth and Weissbluth (1994) have hypothesized that a genetically determined anomaly of the photoneuroendocrine system, modulated by the pineal organ, is the primary source of the Sudden Infant Death Syndrome. We suggest, more specifically, that transient (a few months) ontogenetic dependence of structures within the limbic system upon the antiepileptic properties of melatonin increase the probability that its transient suppression (on the order of 1 to 10 hours) would facilitate the desynchronization of the normal descending pathways from the amygdala and the insula into the vagal complex within the lower medulla (Persinger, 1995). Consequently, sudden suppressions of circulating melatonin, which would encourage this desynchronization, would increase the risk of sudden cardiac death.

Innervation of the pineal organ by sympathetic fibers occurs during the third and fourth months after birth which is the peak period for SIDS. If there is a transient dependence of normal electrical activity of brain-stem structures upon central levels of melatonin, then smaller pineals (Sparks & Hunsaker, 1988) in infants who exhibit SIDS may have decreased capacity to maintain this critical concentration. Transient nonfatal periods of epilepticity could be associated with brief periods of hypoxia, several days to weeks before death that would be sufficient to induce the observed proliferation of microglia within the dentate gyrus (Deligio & Becker, 1994). Petechiae within the lungs, indicative of extravascular blood, is a common correlate of hypoxemia (Naeye, 1976b) and is frequently observed in SIDS.

Although the complete function of melatonin levels within the human infant brain has not been assessed, this serotonin derivative is strongly associated with the acquisition of rhythmic organization of respiration in the

ovine fetus (McMillen, Nowak, Walker, & Young, 1990). If infants who die suddenly also display anomalous carotid sinuses (Naeye, *et al.*, 1976), then normal input (concerning levels of carbon dioxide within the blood) into the area postrema and vagal complex could be compromised. A failing baroreceptor reflex, that could be associated with vomiting or lethargy (attenuated cortical activation) would be expected.

A sudden decrease in melatonin levels would exacerbate these potentially fatal responses. Although the suppressive consequences of white light upon the synthesis of melatonin are conspicuous, the effects of low-intensity, extremely low-frequency magnetic fields are less known (Lerchl, Honaka, & Reiter, 1991). For several years Eckert (1992) has been attempting to isolate the specific parameters from power lines to static geomagnetic anomalies, which could trigger SIDS. Weissbluth and Weissbluth (1994) considered geomagnetic variation as one possible trigger for infants who succumb to the Sudden Infant Death Syndrome.

That increased amplitudes of global geomagnetic activity are associated with lower thresholds for seizure activity has been reported for human and nonhuman species (Persinger, 1995). A threshold effect may require an increased average (daily) amplitude (nT) above about 20 nT. Bureau and Persinger (1995) reported a clear decrease in the onset time of seizures in adult rats following lithium/pilocarpine injections when ambient geomagnetic activity was above 15 nT to 20 nT, while Persinger and Richards (1995) showed specific increases in vestibular experiences in young human normal adults during the days following nights when geomagnetic activity exceeded this value.

We reasoned that a significant proportion of the deaths attributed to SIDS would be associated with specific increases in increments of amplitude of geomagnetic activity. Jacobson's equations (Jacobson & Yamanashi, 1994), if generalizable, suggest that very small intensities within the range of picoT to nanoT could influence brain chemistry. However, we suspect that increments of the magnitude of variations in global geomagnetic activity, which are effectively scalar values, are coupled to the production of specific bands of spectral frequencies. They would be the functional stimuli which interact with neuroelectrical processes. This coupling is analogous to the classical confound between the increased amplitude and the decreased frequency for electroencephalographic phenomena.

Method

We decided to check whether there was a significant correlation between the numbers of cases of SIDS per month and the numbers of days per month exhibiting average aa (average antipodal index; Mayaud, 1973) values between 0–10 nT (nanoTesla or gamma), 11–20 nT, 21–30 nT, . . . to

100 nT (values greater than 100 truncated) of activity for a total of 10 intervals. Although we would have preferred to evaluate daily cases, particularly in light of the propensity for cases of SIDS to cluster in time, the data base was too limited.

The numbers of all cases attributed to SIDS within the province of Ontario for each month for the two years 1960 and 1961 ($M = 11.0$, $SD = 4.2$, range = 4.0 to 17.5) were obtained from Steele, Kraus, and Langworth (1967) because the data were easily accessible. The numbers of days within each of the specific 10-nT increments of average geomagnetic activity (northern hemisphere) were also obtained from Mayaud's (1973) average antipodal (aa) index for each month for 1960 and 1961. Because Steele's monthly data were a composite of both years we computed as well the average for the number of days with specific geomagnetic activity per month for those two years. The means and standard deviations (in parentheses) for numbers of days per month with successive 10-nT increments in aa values were 0 through 10 nT (3.9 and 1.8), 11 through 20 nT (6.9 and 2.6), 21 through 30 nT (7.2 and 2.4), 31 through 40 nT (4.9 and 2.3), and 41 through 50 nT (2.4 and 1.4); all other increments contained values greater than 0.5 but less than 2.0. Despite the range in numbers of days with various increments of aa values, the coefficients of variation were comparable.

To minimize the consequences of obvious dependence between the ten categories (considering the fixed numbers of days per month), the numbers of days within the specific range of geomagnetic values for the 12 months were factor analyzed (PA1 method). This resulted in four factors whose (standardized) scores were employed as independent variables within a multiple regression to predict the mean numbers of cases of SIDS per month. All analyses involved SPSS software on a VAX 4000 computer. Linearity of equations and components was verified by visual inspection of scattergrams.

Results

The results of the multiple regression with the numbers of cases of SIDS per month as the dependent variable and the (four) factor scores for the numbers of days within successive 10-nT increments of daily average global geomagnetic activity as independent variables indicated that only two factors entered the equation, resulting in a multiple R of .91 ($F_{2,9} = 20.54$, $p < .001$; adjusted $R^2 = 78\%$). The first factor (fourth in strength, eigenvalue = 1.20, with 12% of the variance accounted for) was loaded (r values in parentheses) primarily and significantly ($r > .50$) by decreases in the numbers of days with aa values from 21 through 30 nT ($r = -.79$) but increases in the numbers of days with values between 31 and 40 nT ($r = .65$) and 81 through 90 nT ($r = .56$). The second factor (second in strength, eigenvalue = 2.33 with 23% of the variance accounted for) was loaded primarily by decreases in the

numbers of days with values of 11 through 20 nT ($r = -.91$) but increases between 71 and 80 nT ($r = .76$).

The partial regression coefficients for the two factors were 3.31 (beta = 0.78) and −1.92 (beta = −0.46), respectively. The standard error of estimate was 1.98 (the Y-intercept for the equation was, as expected, 11.04). There was no significant correlation between either factor scores or the numbers of cases of SIDS and the sequence of months; the Durbin-Watson value for this time series was 1.96. When accommodating the direction of the partial slopes, this equation indicated that incidents of SIDS occurred within months in which geophysical factors produced more days with daily global aa values of 11 through 20 nT and 31 through 40 nT but fewer days with values of 21 through 30 nT. Partial correlation between the equation, the numbers of cases of SIDS per month and the numbers of days in each month (28 to 31 days), a minor error term that was considered potentially important in light of the size of the multiple R, did not alter the strength of the effects.

To discern if the 10-nT increments had been the optimal increment of analysis (or "window"), 5-nT and 20-nT bins of average intensity were counted. For the first window, the numbers of days per month in which days with values between 0 through 5 nT, 6 through 10 nT, 11 through 15 nT . . . to a maximum of 50 nT (after which all values were truncated to 50) were measured. For the second window the numbers of days per month that display values between 0 and 19 nT, 20 and 39 nT, to a maximum of 100 nT or greater, were obtained. Factor analysis of the numbers of days per month for the 5-nT intervals yielded four factors while the analysis for the 20-nT increments gave two factors.

Only the second (eigenvalue = 1.53, accounting for 19% of the variance) and third factors (1.07, accounting for 13% of the variance) for the 5-nT bins entered the equation to predict (multiple $R = .71$) the numbers of cases of SIDS ($F_{2,9} = 10.60$, $p < .001$; adjusted $R^2 = 50\%$). The strongest factor (partial regression coefficient = 2.88; beta = 0.68) was loaded by an increase in the numbers of days with aa values between 6 and 10 nT (0.50) and 11 and 15 nT (0.70) but decreases in the numbers of days between 21 and 25 nT (−0.96) and 26 and 30 nT (−0.51). The second factor (partial regression coefficient = 1.81; beta = .43) was loaded by an increase in the numbers of days with values between 11 and 15 nT (.56) and 30 and 35 nT (.82). The single most powerful bivariate correlation ($r = -.75$, $p < .01$) occurred between the numbers of days with aa values between 20 and 25 nT and numbers of deaths (slope = −2.94, constant = 21.1; standard error of estimate = 2.91). None of the factors derived from the numbers of days involving 20-nT increments were significantly correlated with the numbers of cases of SIDS.

Discussion

These results support the hypothesis that increases in geomagnetic activity within a specific band or range of intensities during 24-hr. periods could contribute to the neuroelectrical processes which encourage the Sudden Infant Death Syndrome. A general interpretation of the equations which explained approximately 50% to 80% of the variance in monthly incidence of cases of SIDS indicated that deaths were more likely if (primarily) there had been increases in the numbers of days with average global geomagnetic activity between 11 and 15 nT but decreases in days with values between 21 and 30 nT. That these particular increments were not artifacts of the numbers of days per month that contained these ranges was indicated by the absence of significant differences between numbers of days for increments and not strongly correlated with the numbers of cases of SIDS.

Because this was a correlational study, we cannot exclude the possibility that one or more physical, environmental stimuli associated with this particular range of daily geomagnetic activity might be the precipitating factor for the vulnerable organism. For example, sudden death also occurs more frequently four to five days after sudden drops in environmental temperature (Murphy & Campbell, 1987). We suggest that this lag may be typical of the association between increased geomagnetic activity and the intensification of pressure gradients or vortices (Roberts & Olson, 1973) within the troposphere during the formation of air masses (Beynon & Winstanley, 1969; Persinger, 1980).

There may also be specific complex and bioresonant frequencies generated when the perturbations of the steady-state field oscillate through increments of peak-to-peak alterations in geomagnetic amplitude, which we attempted to approximate with the 10-nT increments. If these increments represent real phenomena and not artifacts from a restricted set of numbers (days/month), then these increments of intensity may be associated with the presence of specific resonance frequencies and their harmonics. Although perhaps coincidental, it may be relevant that the Jacobson solution for a synchronous field between 1 and 10 Hz whose amplitude is about 10 through 20 nT would affect a molecular mass within the order of magnitude of approximately 20 kD (a large protein) such as the light-sensitive serotonin-acetyl transferase within the melatonin pathway (Coon, Rosebloom, Baler, Weller, Namboodiri, Koonin, & Klein, 1995).

It is possible that these stimuli are always present but not effective when the infant's neuronal development is beyond this vulnerable stage. Such transient susceptibility would be consistent with the accepted ontogenetic concept of "optimal periods." When these critical geomagnetic stimuli are present, their influence should be greater during the winter months because the melatonin levels are elevated and during the night because a 10- to

50-fold increase occurs in the synthesis of this compound. Sudden depletion of central melatonin by geomagnetic stimuli during the seasonal down-regulation of melatonin receptors within key limbic structures would initiate the potentially fatal electrical paroxysmal activity. Other conditions which decrease birth weight and indirectly prolong the critical period of vulnerability to these stimuli would also increase the risk of producing the conditions that result in the Sudden Infant Death Syndrome.

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